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PROTEIN STIMULATION (LOCAL DEATH) AND PROTEIN THERAPY

"Natural potencies within us are the true healers of diseases."
Hippocrates. Epidemic Diseases, VI, 5, 81.

About 1873–5, over fifty years ago, the great pathologist, Carl Weigert, stated that a local necrosis at some part of a living organism will, as a rule, result in heightened functional activity, usually in excess of requirements of repair, if the initial injury be not too great.[1] This is Weigert's law, or as he termed it, his Siva-theory, after the Hindu god of destruction, whose consort is Kali. In 1880 (Ges. Abhandl., I, 23) Weigert defined this iniatory necrotic activation of tissue repair as a "partial death" (*Partialtod*), an old idea of Bichat's, revamped by Richard Hertwig (1906), with reference to the periodic dissolution of the nucleus and similar regenerative phenomena inside of protozoan cells. Through this view of "local death," as the initiator of a regulative process tending to repair and ultimate healing (necrobiosis), Weigert came into somewhat footless conflict with Virchow, whose theory of a formative stimulus was only another concept of the same thing. If a living organism or process is to be repaired or renewed, something about it has to die. As a phase of divine philosophy, this principle might be generalized to *n* dimensions, from the famous *stirb und werde* of Goethe to the causes of the growth and decay of nations or the laws governing the conservation and dissipation of energy

[1] Weigert: Jahresb. d. schles. Gesellsch. f. vaterl. Cultur 1873, Breslau, 1874, LI, 140. Ges. Abhandl., Berlin, 1906, pt. 1, 12; 16. Pflüger, in 1877, expressed the idea naïvely as follows: "Injury is the incentive to removal of injury."

(maximum entropy) throughout the universe. In 1885, the psychiatrist Richard Arndt, in considering the causation of neurasthenic states, gave, without intention, a new slant to the conditions governing the reparative process by the following generalization: Weak stimuli accelerate vital activities; strong stimuli inhibit them; maximal stimuli suppress or utterly destroy them; or, in plainer English, to overdo is to undo. This generalization, the very essence of the Hippocratic "Healing Power of Nature" (*non nocere*) and implicit, from the start, in the use of poisons as remedies, was then tried out in the pharmacological laboratory upon yeast by Hugo Schulz (1887–1925), and latterly upon plants by Czapek, with the inevitable fatalistic series of consequences, *viz.*, stimulation, paralysis (narcosis), *exitus lethalis*. The principle is now known as the Arndt-Schulz law, but certain glaring exceptions have discredited its universal application, and it is best regarded as a convenient rule or corollary, deriving from Weigert's law. But exceptions often prove a rule, and the validity of the principle has been pretty thoroughly confirmed in such experiments as those of Strangeways and Oakley on Röntgen irradiation of tissue cells *in vitro* (1924), Dalq on the effect of hypnotic solutions on the maturation of sea urchin eggs (1924), Child on regeneration in marine organisms (1924), Haberlandt and Bier on wound-repair in vegetable and human tissues (1921), or Arndt's own subsequent work on the origin of giants and dwarfs and the rationale of degeneration and regeneration (1892–5). In the light of Professor Bier's recent defense of homeopathy (1925), or of the well-known principle that concentration is unfavorable to chemical action (*corpora non agunt nisi soluta*), the Arndt-Schulz "law" acquires some of the commonplace implications of "a self-evident fact stated in a novel manner." It becomes more and more obvious that the real service of Hahnemann to medicine was to stress the *milde Macht* of Goethe, that nature is *maxima in minimis*, that mild remedies and small doses are usually the most efficient.

The whole theory of sensitization of the tissues by the injection or juxtaposition of foreign proteins grew up around the treatment of bacterial infections by specific sera and vaccines. In the early nineties, following the introduction of Koch's tuberculin (1890), Behring's antitoxin (1890) and the vaccines

against typhoid (1888–96), it began to be perceived that, in some cases, such remedies had apparently beneficial effects in other specific infections. Thus, in 1891, Buchner showed that Koch's tuberculin may do some good in other diseases and maintained that its therapeutic effects, if any, are due, not to its specific bacterial derivation, but to the proteins it contains. In 1893, when Fränkel reported on 57 cases of typhoid benefited by injections of Eberth bacilli, Rumpf produced cases benefited by pyocyaneus vaccines. The *furore* over the success of the diphtheria and tetanus antitoxins obliterated these findings, which had also been negatived by other observers. Meanwhile, the tuberculins had been analyzed by Kühne (1891), and in 1895 Matthes and Krehl began to experiment with these protein split products. They found incidentally that reactions, similar to those from tuberculin, may be obtained with other proteins, notably deuteroalbumose and milk. A decade later, Lüdke and Holler were treating typhoid fever on a large scale with intravenous injections of albumose (1915), Schmidt and Saxl had begun the treatment of different infections with parenteral milk injections (1916), Jobling and Peterson were treating typhoid with intravenous proteoses (1916), which were injected with success in arthritis by a number of American experimenters, from Miller and Lusk (1916), to Ralph Pemberton (Army cases, 1920). Before all this had come to pass, Wagner von Jauregg had proposed that general paralysis be treated by intercurrent infections (1887), Terc had tried the effect of bee stings in rheumatism (1888), Fochier had treated pyogenic infections by producing artificial (fixation) abscesses (1891–2), W. B. Coley got striking results with erysipelas toxins in sarcoma (1893), Gilbert had introduced autoserotherapy (1894), Horbacewzki nuclein injections (1894), Petterson the leucocyte extracts (1905–6), Credé collargol and colloidal metals (1906), Beard trypsin therapy in cancer (1911), Schafer the polyvalent brew later commercialized as "phylacogens" (1911), Renaud the treatment of infections with irradiated typhoid vaccines (1911) and Vaughan had experimented with the protein split products (1913).[2] Thus arose

[2] For details of this history see the book on "Protein Therapy and Non-specific Resistance" (New York, 1922) by Dr. William F. Petersen (Chicago), which has been translated into German.

the theory of "non-specific therapy," in contradistinction to orthodox specific therapy with sera and vaccines, the very foundations of which seemed to be shaken by the puzzling fact that the self-same reactions could be produced in the system by injections of protein products, hypertonic saline solutions or even plain water; while most "specific" sera and vaccines were apparently paraspecific (virtually polyvalent) on occasion. When, in 1917–19, Wagner von Jauregg began to treat general paralytics by inoculation of tertian malaria, and Plaut got similar effects by superinfection with relapsing fever (1921), it became apparent that the basic idea was something as old as the hills. For the incompatibility between epilepsy and quartan fever and its disappearances after malarial infection had been known even to the Hippocratic School (Epidemics, vi, 6, 5). Non-specific therapy was, therefore, either "relatively specific," with reference to paraspecific reactions (polyvalence) of individual sera and vaccines, or effected its results by stimulating the defensive mechanisms of the body *via* stimulation of heat-regulating centers (Paltauf, 1915), desensitization (von Groer, 1915), "omnicellular plasma-activation" (Weichardt, 1919), selective stimulation (Döllken, 1919), acceleration and inhibition of inflammation (Starkenstein, 1919) and so on. Whatever the mechanism, local or general, the process has obviously some likeness to the revulsive or alterative effects of such old remedies as bloodletting, the seton, the moxa, acupuncture, vesication, Baunscheidtism (needling), massage, inunction, antimony cups, quassia cups, mineral waters (radioactivity), Junod's haemospasia (giant cupping or bloodletting without letting blood), the alterative effects of mercury, arsenic or iodides, the *bouleversement* in sea sickness, or even H. C. Wood's way of treating a cold: take a violent emetic, then a warm bath, then a champagne and oyster supper in bed.

In the case of non-specific injections, the reaction is always the same, *viz.*, initial chill, fluctuating rise of temperature, increased pulse-rate, temporary decline of blood-pressure, increased leucocytosis, increased glandular activity, accelerated metabolism (nitrogen), increase of enzyme production and of immune substances, with the well-known focal (*Herd*) reaction or inflammatory area at the site of injection. Nausea, vomiting, headache,

delirium and other untoward symptoms are to be regarded as the effects of too liberal dosage. The stimulation of local cellular activity has been attributed to the catabolic products of the injured cells, which Gutherz labels provisionally, necrotines or metabolines,[3] to the breaking down of the parenteral (injected) protein, to change of ion partition (acid-base equilibrium), to disturbance of ferment-antiferment balance (Petersen), and so on. Whether the starting point of these changes be the liver or the whole reticular-endothelial system, the essence of the process is an indirect stimulation or revulsion. In the face of a certain percentage of striking results in typhoid fever, the arthritides, gonorrhœa, the communicable diseases, asthma, neurosyphilis, diseases of skin and the eye, there have been many fatalities, particularly in malarial superinfection against neurosyphilis and in the use of krysolgan and sanocrysin in phthisis. In the view of prominent German hospital clinicians, the method, if pursued haphazard by private practitioners, is fraught with the danger of reducing the patient to the level of an experimental laboratory animal, and in actual laboratory experimentation there have been salient and significant discrepancies. There is, to begin with, no agreement whatever between the animal and human findings. After parenteral protein injections, guinea pigs fever readily, dogs and humans moderately, rabbits little, if at all. There is marked difference in animal and human reactions in diphtheria, typhoid and cholera. Results are dubious in tuberculosis, negative in tetanus. The inflammatory reaction is diphasic or Janus-faced (Wolff-Eisner). The focal reaction of tuberculin may be produced by a variety of agents, while many non-tuberculous processes respond to tuberculin. Toxic doses of foreign proteins are always dangerous, often fatal, and the correct threshold dose is difficult to estimate offhand and varies in different individuals. The method collapses wherever one man's meat may be another man's poison. It takes two to make a cure, the patient as well as the remedy. The whole duty of the bedside practitioner is not to make a *Versuchsthier* of the patient, but, in the words of Trudeau, "to heal sometimes, to alleviate often, to comfort always." In this

[3] S. Gutherz: Der Partialtod, Jena, 1926, *passim.*

view, protein therapy, in its present tentative phase, acquires the polychrome, multifarious aspect of the older pharmacopœias or of the vagaries of homeopathy. Polypharmacy and panaceas are still liable to the sarcasm of the old Halle clinician: "We seem to be standing before the booth of a mountebank." The merits of protein therapy, however, are its possibilities of extreme delicacy in plan and execution. Gordonoff activated a stopped heart with a single drop of chlorophyll (1925–6). Loeb activated the unfertilized ovum to parthenogenesis with nucleic acid. It is said that a deviation in the hydrogen-ion concentration of the blood no greater than that between tap water and distilled water is fatal. Injection of haematoporphyrin in man is so potent in its photodynamic effect that it may kill even on a cloudy day. Freund found that injection of a rabbit's own defibrinated blood into the animal directly after coagulation is invariably fatal (1921). A quarter of an hour later, the result is different. Walbum found that manganese is specific for staphylococcus infection in rabbits; that mice poisoned with ratin may be immunized by caesium and iridium in certain concentrations; while aluminium and manganese are most effective in tuberculous guinea-pigs (1925). Bier, in his enthusiasms over Hahnemann, got isopathic effects by small ether injections in ether-bronchitis and latterly in other bronchitides. The efficiency of sulphur, in boils and cutaneous staphylomycoses, he attributes to its capacity to stimulate the skin to repair its own troubles. Schulz has evolved a special pharmacotherapy based upon minute dosage. Protein therapy, like specificity in dietetics, is yet in its infancy. The crux of the matter is the specific reaction of the individual patient to the threshold dose and here the Bichat-Weigert principle, with the Arndt-Schulz corollary, may serve as a guiding principle through this particular maze of modern medicine.

F. H. Garrison

PAPERS DELIVERED AT STATED MEETINGS

THE PATHOLOGY AND NEUROLOGIC SYMPTOMS IN PERNICIOUS ANEMIA

(*Abstract*)

ISRAEL STRAUSS

(Delivered before The New York Academy of Medicine, April 21, 1927)

The pathologic process which is observed in the central nervous system in cases of pernicious anemia is a degenerative one. It consists essentially in a demyelinization of nerve fibers associated with or resulting in a breaking down of the axones. The process commences in small foci which later coalesce, and results in secondary degeneration of the fiber tracts. The first site of the process is usually either the lumbo-sacral segments or the upper cervical segments of the cord. Later on the middle cervical and dorsal segments are affected. The posterior columns and lateral columns of the cord are most affected. The direct cerebellar tract is likewise involved. It is rare that the gray matter in the cord shows any material affection.

Pathologic changes have been found in the brain. They consist of areas of rarification, foci demyelinization, and of slight changes in the ganglion cells. They are not as extensive or as pronounced as are the changes in the spinal cord. These changes are believed to be due to the influence of some toxin which makes itself felt through the circulation. They are not secondary to changes in blood vessels. It is unknown why the process selects the region of the cord above mentioned so exclusively.

Pernicious anemia is not the only disease in which this pathologic process occurs. It is found in other anemias, which are classified as secondary. It also occurs in the anemia produced by *Bothriocephalus latus*. Occasionally severe and prolonged cachectic states due to carcinoma produce the above changes. They have also been described in leukemia, although here the leukemic infiltration of the blood-vessels may be a factor in their production. It is not infrequently found in diabetes. It has been

described in cases of chronic alcoholism and has been observed in pellagra. Therefore neither the process nor the symptoms arising can be regarded as diagnostic of pernicious anemia.

The neurologic symptoms are such as would be expected from interference in function of the parts involved:

I. Posterior Column Symptoms:
   1. Atony.
   2. Loss or diminution of tendon reflexes.
   3. Ataxia.
   4. Disturbance in vibratory and joint sense.
   5. Parasthesiae.
   6. Girdle sensation.
   7. Bladder.

II. Lateral Column Symptoms:
   1. Increased tonus—spasticity.
   2. Increased reflexes.
   3. Pathologic reflexes (*a*) Babinski.
      (*b*) Clonus.
      (*c*) Loss of abdominal reflexes.
   4. Motor weakness—paralysis.

III. Posterior and Lateral Column Symptoms:

Depends upon the level at which they are most affected, the time in the process when they are involved and the extent of their affection.

Example:

1. Spastic—ataxic paraplegia.
   Clinical picture of pyramidal tract plus ataxia.
2. Tabes syndrome plus motor weakness rigidity plus pathological reflexes of pyramidal tract (Babinski-clonus).

Early symptoms are usually parasthesiae in the extremities and the tongue, ataxia of the lower extremities and disturbance of vibratory sensibility. The symptoms may antedate the appearance of the typical blood picture of pernicious anemia for a period extending from months to years. There may be remissions of symptoms in the early stages. The weakness in the extremities is very frequently a pronounced symptom. This may go on to a paresis which compels the patient to be bedridden. Under treatment this weakness may improve considerably. When, however, the degenerative process in the spinal cord is far advanced, there will be no change in the objective neurologic symptoms even

though the blood shows only slight changes. The same is true in regard to those cases where the condition of the blood has been improved by treatment. Minor and Murphy, in a recent article on the influence of liver diet, showed that the blood had become normal and that the neurologic symptoms had not improved. It is said that the neurologic symptoms occur in from 50 per cent. to 75 per cent. of the cases. They are very frequently overlooked because the physician is not on his guard to detect their early appearance or is not familiar with the technique of neurological examination. Occasionally no neurologic symptoms have been observed and yet microscopic examination of the cord has shown that the changes are present.

Mental symptoms in pernicious anemia occur occasionally, and the clinical picture is said by some to be characteristic of the disease. They are, however, not of frequent occurrence.

COMMENT ON TROPICAL DISEASES OF INTEREST IN NEW YORK CITY—A BRIEF REVIEW

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(Delivered before The New York Academy of Medicine, May 5, 1927)

Diagnosis, under all circumstances, is admittedly a difficult art, and the practitioner in the City of New York cannot well afford to dismiss from all consideration the entire list of diseases which are called tropical because he may deem them foreign to his practical interest.

"Tropical diseases" is a more or less elastic term. It does not imply diseases which are strictly peculiar to the tropics, nor could it possibly mean all diseases which prevail in the tropics. Few diseases are strictly confined to tropical latitudes, and the practitioner in the tropics in his daily life finds himself confronted for the most part with problems very similar to those which confront the practitioner in more temperate latitudes.

The term in general is taken to include those diseases which from one circumstance or another are especially prevalent in warm climates.

When one takes into consideration, however, the large population group which comprises New York City, with its admixture of foreign elements, and its constant and varied connections through the shipping of the port with all parts of the world, the possibility of encountering certain tropical diseases is almost inevitable. With the development of ease and facility in transportation, this becomes all the more likely, especially in view of the fact that the southern United States itself is at least subtropical, and furnishes not a few of the well-known tropical diseases.

It is not altogether easy to determine just which of these diseases may be important in New York City. Mortality statistics are of little help, and there do not appear to be any published hospital statistics of special value in this connection. However, from the opinions of men with wide hospital experience in the city, and the judgment of certain writers on tropical medicine, we may, I think, make a fair inference as to the tropical diseases which the practitioner in this latitude is most likely to encounter and with which he should have some familiarity.

The modern developments of medicine make such a heavy demand upon the time of the practitioner that he is naturally inclined to discard as of little practical interest those diseases which he feels are unlikely to be met in his daily work. Moreover, tropical medicine with its numerous parasitic affections leads into the somewhat complicated field of medical zoology, the by-paths of which appear uninviting and time-consuming.

It is unlikely that with all the resources of a good hospital the diagnosis of tropical disease would long be overlooked, but, to the practitioner who cannot always avail himself of such facilities, the lack of such knowledge may lead to grave errors in diagnosis; and indeed instances are not wanting where such errors have occurred even in good hospitals.

In diagnosis, it is a safe rule always to look for the more common thing rather than the unusual, and it is an equally safe rule to inquire with some care into the history of a patient before venturing a diagnosis. It would certainly be unsafe in most cases to risk a diagnosis of some tropical affection in the person of an individual who had not visited warm climates.

It may be mentioned that the Public Health Service carries on in this and other ports of entry three important functions,

namely, quarantine examination of all vessels arriving from foreign ports, the medical examination of arriving alien immigrants, and the medical care and treatment of a large number of seamen of the American merchant marine. An officer engaged in these duties necessarily comes into contact not infrequently with tropical disease.

In the examination of vessels, the diseases classed as quarantinable are cholera, yellow fever, smallpox, typhus fever, leprosy and plague. These diseases may for the most part be classed as tropical, and since no quarantine is absolute and could not be so made except at prohibitive cost, there is always the possibility of the introduction into the port of quarantinable disease.

The prompt diagnosis and report of such under the circumstances would then be a service of great value. On the other hand, the mistaken diagnosis of quarantinable disease may sometimes be a distinct disservice, since, after report, it will appear on the bills of health issued to vessels sailing from this port and may result in their unnecessary quarantine detention at port of arrival.

My purpose, then, is to direct attention to the fact that certain tropical diseases may not be foreign to the practical interest of the medical man in temperate climates, especially if he is located in a large port; and that to carry "in the back of one's head" some working diagnostic knowledge of the more common of these diseases may at times prove very useful to one's self and perhaps beneficial to one's patients.

With this end in view, I am submitting a brief comment on certain salient features of those tropical diseases believed to be most commonly met with here.

Among the protozoal infections there are two of importance, both of which are really endemic in this state. These are *malaria* and *amebiasis.*

It may seem strange to discuss malaria, but nothing has so impressed me as the inability of a doctor to appreciate the gravity which this disease may at times assume, if he has had practical experience only with its milder manifestations commonly seen in temperate climates.

An experienced officer of the Public Health Service reported some years ago the boarding, in quarantine work off the coast of

Florida, of a small schooner from the West Indies. Among the crew of eight men, six lay dead, stricken at their daily task, and the other two were so desperately ill that they had barely survived to navigate the vessel to port through calm weather. This was malaria in its most malignant form.

The milder benign tertian and quartan infections seen in temperate climates give no indication as a rule of the phenomena observed in infections with the malignant tertian parasite which are so common in warm climates, and fatal errors in judgment are all the more distressing in a disease for which we possess a ready means of diagnosis and a specific remedy.

It should be recalled that visitors to warm climates may acquire an infection and show no evidence of it till their return to temperate latitudes, when they will at once develop a malarial attack. This is a common history.

It must also not be forgotten that severe manifestations of malaria do not permit any expectant plan of treatment. They demand immediate attention and prompt administration of quinine.

In symptomatology, one must recall also that tropical malaria may be totally wanting in the periodic chill, fever and sweat. It may be acute or chronic, atypical, in many ways simulating a variety of diseases, and above all it may suddenly assume a pernicious type of the utmost gravity.

The disease has the property of latency, and infections acquired in warm climates may not show activity for a long period of time. This suggests the caution also that the finding of parasites in the blood is no confirmation that the active disease from which the patient may be suffering is necessarily malaria.

The cardinal points in diagnosis are fever, which usually displays a certain intermittency in its course and a certain periodicity in its recurrence, anemia, enlarged spleen and parasites in the blood. Parasites are at times difficult to find, especially if quinine has been administered. It may be added that any fever which resists quinine in full doses for two days or more is not likely malaria.

As for treatment, in severe manifestations the administration of quinine by the intramuscular or intravenous route must be considered. In the treatment of any case of malaria, especially

in view of possible relapses, one should not forget the standard of the National Malaria Committee, which advises for the acute attack 10 grains of quinine sulphate by mouth three times a day for a period of at least three or four days, to be followed by 10 grains each night before retiring, for a period of eight weeks.

Finally, when we possess a reliable and simple method of accurate diagnosis, it would seem a slovenly habit to prescribe quinine without first making use of that method, and it is a habit not without danger.

Although rarely seen here, mention should be made of a condition known as Blackwater fever, which, though of disputed etiology, has of late years by common consent been accepted as a manifestation of malaria. It is more common in those who have spent some time in malarious regions and have suffered several attacks of malaria. The integrity of the red blood-cells becomes impaired, and following a dose of quinine, undue fatigue, too much exposure to the sun, or a chilling of the body surface, there occurs extensive lysis of the red cells, accompanied by a prostrating chill, early jaundice and the passage of porter-colored urine—hemoglobinuria.

The condition is a serious one, and although of malarious nature, the administration of quinine is not always advisable. The remedy must be used with caution backed by nice judgment.

The other protozoal infection mentioned—amebiasis—gives rise to two clinical conditions of so much importance that usually they are described separately as amebic dysentery and amebic abscess of the liver. These are, however, only incidental in the evolution of the infection.

There are several varieties of ameba which occur in the intestinal tract of man as harmless commensals. The pathogenic variety is the *Entameba histolytica*. The infection occurs primarily in the intestinal tract. The normal habitat of the organism is the mucous membrane of the large intestine. It is a true parasite and lives at the expense of its host. In most cases a biologic equilibrium is established between host and parasite and the host becomes simply a carrier, without apparent disturbance of his health. In the carrier state, the organism is discharged in its encysted, infectious form.

If the equilibrium is disturbed, the parasites damage or destroy the tissues to such an extent as to produce pathologic effects, and the illness takes the form of an ulcerative colitis. The patient then suffers from amebic dysentery, or under more favorable circumstances, simply an amebic diarrhea.

Under unfavorable circumstances, the ameba through the portal circulation may invade the liver, where it produces inflammation and perhaps ultimately abscess formation. Rarely the organism may go further and produce abscess of the lung or brain, or involve other tissues.

It is to be noted that the carrier discharges the encysted form of the parasite, which is the infectious form, and the carrier state is responsible for the transmission and propagation of the infection.

The condition known as amebic dysentery is an important member of the dysentery group. The term dysentery designates a syndrome of small, frequent mucous or muco-sanguinolent stools accompanied by tenesmus and griping pains. The classification of the dysenteries is based largely on etiology. Amebic and bacillary dysentery comprise the two important members of the group.

In diagnosis it may be recalled that amebic dysentery is distinguished from the bacillary form by more insidious development, greater chronicity and, as a rule, an afebrile course. Ultimate diagnosis must be established by the demonstration under the microscope of the *Entameba histolytica*.

In the treatment of amebic dysentery, ipecac in one form or another has for a long while held an important place, and deservedly so. It may be added that ipecac is not a harmless remedy, and for the avoidance of serious and unpleasant consequences it must be used with discrimination.

The other important episode in the evolution of amebiasis is abscess of the liver, a grave and not infrequent condition, which in the hands of even experienced clinicians is often either overlooked or mistakenly diagnosed.

The clinical picture is characterized by insidiousness, with or without previous history of dysentery, more or less indefinite local signs and symptoms, septic fever, loss of flesh, a curious, yellow, muddy tinge to the skin, coated tongue, general listlessness, moderate leucocytosis and a prolonged and chronic course.

There is enlargement of the liver, which, if it occur upward, may be detected by the X-ray. Diagnosis may be confirmed by aspiration of the liver, which should not be too much delayed. Unlike aspiration of the spleen, it is a comparatively safe and harmless procedure. The appearance of the pus is more or less distinctive. If pus is found, it must be drained at once.

In treatment, mortality from the open operation is high. It has been greatly reduced by a coincident treatment with emetine.

Beri-beri, pellagra and *sprue* form another tropical group traditionally classed together by reason of an alleged etiologic relationship to faulty diet, which relationship in the minds of many is not yet determined.

Beri-beri is especially common among the rice-eating peoples of the far east. It is a multiple neuritis involving not only the nerves of the extremities but also the vagus and vasomotor nerves.

Clinically it appears in several forms, a wet or dropsical form, a dry or paraplegic type, or a mixed type. The involvement of the vagus makes sudden death from acute failure of the heart one of its striking characteristics. Cardiac disturbance with fatal tendency may develop suddenly in the mildest cases. There is naturally great variation in symptomatology.

In beri-beri literature the term "ship beri-beri" is applied to the disease developing among crews of vessels, usually during a long voyage, and most commonly on sailing vessels. This condition is believed by some to be more closely related to scurvy in that more prompt recovery is said to take place when the patients are placed on a full, balanced ration, and it is also stated that there is less degenerative involvement of the nerves.

There is also an infantile beri-beri which in the orient is of importance.

Pellagra is very common in the southern United States, but cases are uncommon in New York City. It is unlikely that such cases would be diagnosed in the absence of the characteristic skin eruption which is seen in this disease, although the disease may exist without this distinctive skin lesion.

The skin rash of pellagra is pathognomonic and is characterized generally by the occurrence of an erythema on exposed body surfaces, most frequently on the backs of the hands and wrists, the face and the back of the neck. The eruption is characterized by

its symmetry, sharply defined borders and its color, which, at first, is red like a sunburn but later assumes a violaceous tint and finally a deep sepia brown. This eruption goes through a regular process of evolution which lasts a few weeks. With the eruption, as a rule, will be found gastro-intestinal and nervous disturbances. The gastro-intestinal disturbances will be sore mouth, indigestion and diarrhea, and the nervous manifestations are varied, chiefly depression, anxiety, sleeplessness and hyperesthesiae.

Sprue, or tropical sprue, is not infrequently seen. It is a peculiar and dangerous form of chronic catarrhal inflammation of the whole or part of the mucous membrance of the alimentary canal. Although a disease of warm climates, it may develop for the first time in temperate climates in individuals who have previously resided in warm climates.

Its diagnosis rests first upon the peculiar condition of the tongue, which shows superficial erosions, patches of congestion, minute vesicles and a general redness with frequently the appearance of fissures. This condition is also noted on the general mucous membrane of the mouth. This is accompanied by dyspeptic distension of the abdomen and a morning diarrhea with rather characteristic stools which are semi-solid, yeasty, copious and lacking in color.

Among the important bacterial diseases may be mentioned *tularemia, Malta fever,* and possibly *plague.*

Tularemia is a new disease and is classed by writers on tropical medicine among tropical diseases. Its description is just beginning to find its way into the text-books.

Its prevalence is, of course, unknown, but cases have now been found quite widely in the United States. It is due to the bacillus *tularense* and, like plague, seems to be primarily a disease of rodents, conveyed to man by an infected blood-sucking fly or tick. The rodents which have been most responsible for its transmission are wild rabbits and hares, although it occurs in ground-squirrels and wild rats.

It is striking that a number of cases have occurred among laboratory workers handling cultures of the organism or animals infected with the same, the means of the transmission not being thoroughly understood.

Clinically there are recognized four types, the ulceroglandular, the oculoglandular, the glandular and the typhoidal. In the ulceroglandular and oculoglandular, the site of the inoculation is obvious. In the glandular type and in the typhoidal type, the point of entrance of the infection is not apparent.

The onset is sudden with headache, vomiting, chilliness, bodily aching and prostration. The lymph glands draining the region of the point of inoculation are enlarged, tender and frequently proceed to suppuration. There is a continuous fever, with some remissions, for two or three weeks and a moderate leucocytosis. Sometimes a skin eruption is noted which is macular, papular or pustular. Convalescence is slow. Recovery usually takes place and death is rare.

The diagnosis depends in large measure upon the history of the case, with the assistance of a primary lesion on the skin or in the eye, persistent glandular enlargement, fever, and the development in the patient's serum of an agglutination reaction to the specific organism during the second week of the illness. There is no specific treatment.

Malta, or undulant, fever, in accordance with good observers, may be encountered now in almost any part of the United States.

An endemic focus of the disease exists in the southwestern part of the United States. Moreover, recently evidence is not lacking that an organism, *Brucella abortus*, which is the cause of contagious abortion in cattle, is very closely related to, if it be not a possible variety of, the organism which causes Malta fever, *Brucella militensis*. Cases which symptomatologically coincide with Malta fever have been reported in the United States, due to infection with the *Brucella abortus*. Clinically, therefore, in the literature we now see descriptions of infections of the *militensis-abortus* group. This has raised a question as to whether the disease may not be spread in cow's milk as well as by its usual mode of transmission—the milk of goats.

Malta fever like any other infection may, of course, assume mild or severe forms. Its mortality is not high but it is a prolonged disease with a very slow convalescence. In its most common form it is characterized by marked waves of intermittent fever of variable length and intensity, separated from one another by periods of temporary absence of symptoms. It is a disease

characterized by a series of febrile attacks with accompanying phenomena. These attacks may be repeated many times and the disease may be much prolonged. As stated, convalescence is slow.

The organism of Malta fever offers peculiar hazards to laboratory workers, and infection from this organism is not uncommon among those who handle it in their daily work.

There is no specific treatment for the disease. Vaccines have been tried but have not given brilliant results, although their use is reported to be of some advantage.

It is unlikely that a case of plague would come under observation, but, since during the past few years this disease has become very widespread over the earth's surface, it may be mentioned as rather exceptional that a port having such world-wide connections as New York City should have escaped this infection. There is a belief among sanitarians that for some reason, not fully understood, this disease will not spread in the port of New York, due possibly to some peculiar circumstance in the life history of the flea through which the disease is conveyed from rodents to man. Nevertheless, it would seem wise for any practitioner in a port of this size and character to bear in mind the possibility that he may some time meet with plague, most likely in its bubonic form, very unlikely in its pneumonic form. It may be added that the disease is endemic among the ground-squirrels of the Pacific Coast and human outbreaks have occurred on that coast.

There are two spirochaetal diseases which may be mentioned. These are *yellow fever* and *Weil's disease,* or infectious jaundice, neither of which is likely to be encountered in this city. There is a third disease, the etiologic factor of which is unknown, but which has been shown to be spread by certain species of mosquito, which, with more likelihood, might possibly be met with. This is *dengue.* This disease during the summer months is not uncommon in the southern United States, and during its prevalence persons may readily travel to this city during its incubation period, developing the disease in the city. It presents an acute picture of rather brief duration with practically no mortality. Its invasion is characterized by a chill or chilliness with fever, severe pain in the back and limbs, a rather typical facies and the later appearance of a polymorphous eruption. A marked leucopenia takes place in the first three or four days of the illness. The

facies of this disease is worthy of comment since it resembles closely that of yellow fever, and to a lesser extent that of the measles.

The four striking clinical characteristics of yellow fever are facies, early jaundice, early albuminuria, and later the want of correlation between temperature and pulse. The last three signs do not appear in dengue.

Typhus fever, the etiology of which is unknown, but which is known to be conveyed by the louse, would seem a disease of some importance to the practitioner of this city. It will be recalled that some years ago, Dr. Brill, of this city, called attention to a typhus-like disease which was endemic in New York and reported a large number of cases. This disease crept into the literature as Brill's Disease.

It may also be recalled that there is a well-known type of typhus fever which occurs in Mexico and goes by the name of tabardillo. Besides this, we have the European form of typhus.

There is a unanimity of opinion that all three of these diseases etiologically are the same disease, but in spite of this Brill's disease continues to preserve a sort of identity of its own, being very much milder in its manifestations, and differing somewhat from the others in its epidemiology.

For practical purposes, the practitioner is unlikely to meet with any form of this affection, except Brill's disease, and this may be encountered not infrequently. It is characterized by a temperature of from two or three weeks' duration and a rather distinctive rash. Its mortality is low and there is no specific treatment.

It is of interest to know that there is another disease endemic in the United States, the symptomatology of which closely conforms to that of typhus fever, but the etiology of which, though unknown, must differ because it is conveyed by a biting tick. This disease prevails rather extensively in the northwestern United States and is known as *Rocky Mountain spotted fever.* In some sections it is highly fatal.

Among the helminthic infections there is one of rather common occurrence, namely *uncinariasis,* or *hookworm.* This disease has been so much studied and so widely written about that it seems unnecessary to do more than simply call attention to it. It is a

chronic infection, very common in the southern United States and, of course, might be encountered at any time. Clinically it is characterized by anemia and attendant symptomatology and its diagnosis rests upon finding the ova in the stools. Treatment is effective if applied with judgment and persistence. There is an old world hookworm and an American species, which are distinct, but the symptomatology is the same.

As of possible interest it may be mentioned that there is another helminthic infection which is very common among Chinese immigrants. This is *clonorchiasis* or *liver-fluke* disease. The interest in this disease lies in the fact that a great many Chinese who come to this country are infected with this worm. For the most part, however, they have no symptoms of disease, the diagnosis being made by finding the ova in the stools. The question of importance has been whether such immigrants should be admitted to the United States, since, according to its life history, the parasite must undergo some phase of its development in a species of fish, so far unknown in this country. The regulations require at the present time that such cases should be deported, but under the circumstances and conditions the spread of the disease in this country would appear a remote possibility.

There is one disease of unknown etiology, called *climatic bubo*, which may be of some significance. These patients present a chronic inflammatory condition of the inguinal glands, obviously not due to any of the known venereal diseases, accompanied by a low-grade fever, with considerable periglandular inflammation, a prolonged course, and in about one-half the cases suppuration of the glands.

This infection is quite common in the West Indies and has for practical reasons received much attention from the medical officers of the United States Navy.

Ordinarily its etiology is regarded as unknown, but some naval medical writers are inclined to think that it may be venereal in its origin, and may represent some new venereal infection. Surgical treatment is not very satisfactory. The injection of foreign proteins and the use of antimony-potassium-tartrate intravenously have been highly recommended.

In conclusion, I may say that I have purposely omitted any consideration of tropical skin diseases, not a few of which may

be met with in the skin clinics of this city. Leprosy is, of course, fairly common in New York City and may be seen at many of the clinics, since the sanitary laws do not necessarily require isolation here. Yaws is very rarely encountered. There is an endemic focus of filariasis in South Carolina, but I am under the impression that the disease is seldom seen here. I might add that in patients from warmer latitudes various intestinal parasites are not uncommon and a microscopic examination of the stools of these individuals will often display evidence of them.

I have made no references to literature, but my indebtedness to standard literature is obvious, particularly the excellent manuals of Stitt, Manson, and Castellani and Chalmers, as well as the more extensive system of Archibald and Byam.

THE THYROID GLAND AND ITS RELATION TO DISEASE

DAVID MARINE

(Delivered before The New York Academy of Medicine, May 19, 1927)

There is no field of medicine where greater confusion and more nonsense exist than in connection with the real and fancied relations of the thyroid to various clinical syndromes on the one hand and the relations of these several clinical syndromes to structural alterations in the thyroid on the other hand. The truth of this statement is clearly demonstrated in the numerous classifications both clinical and anatomical that have arisen, particularly involving Graves's disease.

Regarding the relation of the thyroid to clinical disease there are many reasons and some justification for the formidable array of clinical terms now in use. The most important ones, however, depend on the highly variable symptomatology and on the fact that we know almost nothing regarding the nature of Graves's disease.

Regarding the relation of structural alterations in the thyroid to clinical manifestations there is no justification for the present

confusion. This confusion is due largely to the attempts to correlate clinical diseases believed to be due to the thyroid with structural alterations in the gland. This is quite impossible. Virchow pointed this out more than sixty years ago in the following words: "With reference to true goiter the opinion has long been held that it comprises a series of definite varieties (struma hyperplastica, colloides, cystica, ossea, vasculosa, hemorrhagica, nodosa, etc.) which can develop independently of each other. This is wrong. All these so-called varieties are only different modes of development of essentially the same form of goiter . . . and a large and striking variety of these metamorphoses may exist in the same goiter." Theodor Kocher in 1902 pointed this out in connection with Graves's disease and my own studies are in entire agreement with these observers. We believe that the thyroid has but one cycle of morphological changes. This cycle occurs in all animals with the ductless thyroid and has no necessary relation to the various clinical associations.

As regards the clinical classification of thyroid diseases even greater confusion exists. In addition to the purely clinical terms like simple, endemic, sporadic, physiologic, nodular, adolescent, toxic, hyperthyroidism, thyreotoxicosis, toxic adenoma, dysthyroidism and a host of others, we have still another group of terms—truly a grotesque admixture of clinical and anatomical conceptions—as, for example, toxic non-hyperplastic, toxic hyperplastic, non-toxic hyperplastic, non-hyperplastic non-toxic, etc. These particular terms were introduced by workers at the Mayo Clinic in order to evade designating any case as Graves's disease in which the thyroid was not actively hyperplastic. They vigorously supported this view for several years, which is summarized in their often-quoted published statement that given the condition of the thyroid one could predict the symptomatology and *vice versa*. Such a position was of course untenable.

If we disregard all the fictitious and artificial classifications that have been proposed and base our conception of the relation of the thyroid to disease on what is definitely known regarding the physiology, the chemistry and pathology of this gland, we would at least be on firm ground, although I am sorry to say our present knowledge falls far short of any definite understanding of the subject.

Basing our conception of thyroid diseases, therefore, on the physiology, chemistry and pathology of the gland, we have come to the following classification:

I. Thyroid insufficiencies—
   (*a*) Simple goiter (endemic, sporadic)
   (*b*) Myxedema
      (1) Infantile (cretinism)
      (2) Adult (Gull's disease)

II. Graves's disease.

In my opinion Graves's disease does not properly belong with the true thyroid diseases, but since the thyroid is an important factor in it and we lack the knowledge necessary for a more accurate classification, it is best to leave it here for the present.

Brief reference may be made to certain of the clinical features of thyroid diseases.

With reference to the thyroid insufficiencies the literature of the last ten years indicates that there are now no great differences of opinion regarding the essential features of simple or endemic goiter and myxedema, although quite within the memory of many of us the same confusion existed as exists to-day in relation to Graves's disease. Every one, I think, agrees that simple goiter is a work hypertrophy of the thyroid and is dependent immediately upon an absolute or a relative insufficiency of iodine. This of course leaves open the question of the remote, predisposing and possibly fundamental cause of the disease. It is now known that the thyroid will enlarge when the intake of iodine is below the normal body needs or when the body needs for one reason or another are so increased that the normal intake is for the time being insufficient. The former condition is seen more particularly in the great goiter districts of the world. The latter is seen in the so-called sporadic goiter occurring in association with various infectious diseases, pregnancy, abnormal diets and certain derangements of internal secretion (status lymphaticus, Addison's disease, acromegaly). Further, there is a general agreement both among internists and surgeons regarding the means of prevention and the limitations of treatment. No one would claim, however, that we are near a final understanding of this subject, and I have referred to simple goiter and myxedema mainly to

emphasize how a few facts, if they are crucial ones, can destroy concepts based on ignorance.

In contrast with simple goiter we know practically nothing regarding the fundamental disturbance underlying Graves's disease. It occurs spontaneously only in man, and this deprives us of one important means of approach which so materially aided us in simple goiter. Up to the present time it has not been completely reproduced in animals although there are suggestive studies in this direction. There are two prevailing theories: (1) That the disease is of thyroid origin, and (2) that the disease primarily depends upon a constitutional anomaly or defect which renders the individual's autonomic nervous control over various visceral functions susceptible of injury by a great variety of nonspecific influences, such as fright, worry, over-work, the undermining of nutrition by infectious disease, etc. This constitutional defect may be congenital or acquired and appears to be closely related to that present in status lymphaticus and Addison's disease. Experimentally a symptom-complex related to Graves's disease can be produced in rabbits and cats by sufficient but sublethal injury of the suprarenal glands. In rabbits we have been able to obtain a definite increase in the metabolic rate, beginning from three to six days after suprarenalectomy and lasting from a week to several months, provided the thyroid gland is intact. This effect we have attributed to insufficiency of the cortex. During this reaction there is thymus regeneration which is also characteristic of Graves's disease. Such animals are hypersusceptible to a great variety of physical and chemical agents, just as in the case of Graves's disease. The well-known Goetsch test is an example of this hypersusceptibility, and while I do not believe that such a test is specific for or diagnostic of Graves's disease, it is highly probable that individuals manifesting hypersusceptibility to adrenalin have the constitutional anomaly or defect which underlies the development of Graves's disease.

Finally, I believe it is necessary to discard the view which seems to have gained wide acceptance in recent years, namely, that Graves's disease may be split up into several clinical entities, as for example, "exophthalmic goiter," "adenomatous goiter with hyperthyroidism" and "toxic adenoma." The protean symptomatology of Graves's disease has been emphasized for more than

fifty years, and from what we know of the disease it seems more rational to assume that the variations seen are dependent upon the variations in the activity of the visceral nervous system with the thyroid function remaining qualitatively unchanged, than to assume that all these clinical variations are dependent upon either qualitative or quantitative changes in the thyroid gland.

Since there is no morphological basis in the thyroid for thus splitting up Graves's disease, certain observers, particularly Plummer, have attempted to separate toxic adenoma from Graves's disease by means of the iodine reaction. His hypothesis assumes that iodine causes a drop in the metabolic rate in exophthalmic goiter because iodine completes an incomplete thyroxine, whereas iodine, according to him, does not lower the metabolic rate in case of so-called toxic adenoma. This hypothesis does not include all the facts. In the first instance a great many cases of Graves's disease with adenoma react with iodine with a temporary fall in metabolism, and in the second instance not all cases of Graves's disease without adenoma react with iodine. The phenomenon may have a simpler explanation than that advanced by Plummer, in that the fall in metabolism might be dependent upon the well-known involuting effect of iodine on hyperplasia. Iodine administration causes a rapid storage both of iodine and colloid in the follicles and a rapid involution of the hyperplasia. The stretching and distention of the follicles with colloid causes partial occlusion of the perialveolar capillaries and lymphatics and a pressure retention which temporarily blocks excretion until the thyroid cells have accommodated themselves to the increased pressure and until the vascular capacity of the alveolar capillaries is restored. In support of this view are the following facts: (1) Iodine administration does not lower the increased metabolism caused by feeding desiccated thyroid; (2) iodine does not materially change the metabolic rate in individuals with fully involuted thyroids; (3) iodine when given in small doses does not cause a rapid storage of colloid or a rapid fall in heat production; (4) the fall in heat production corresponds in time and probably in degree with the morphologic effects of involution of the hyperplasia; (5) desiccated thyroid does not cause a rapid fall in metabolism even in cases with hyperplastic glands; (6) cases with adenomatous hyperplasia which do not react to iodine

with storage and involution do not show a lowering in metabolism. The same temporary drop in metabolism can be produced in animals provided the gland is hyperplastic and large doses of iodine are used, and Martin has recently shown that a drop in heat production can also occur in simple goiter in man. The explanation, therefore, of the temporary drop in heat production seems to be that the sudden administration of large amounts of iodine causes a rapid storage of colloid with distention of the follicles, which in turn produces a pressure retention or inhibition of the secretory activity. Within about two weeks, however, the cells accustom themselves to this alteration and the metabolism rises again. The phenomenon erroneously known as "iodine thryroiditis" is an excellent clinical illustration of the effect of the sudden administration of large amounts of iodine to individuals with hyperplastic glands. The dangers that accompany the uncontrolled use of iodine in individuals with the anlage of Graves's disease are serious, and I hope Dr. Goetsch will emphasize this feature.

Summary

The thyroid gland appears to have but one morphological and physiological cycle, and it tends to repeat this cycle in response to stimuli for increased or decreased activity. The morphological and chemical changes observed are, therefore, not specific for any particular clinical syndrome.

There is sufficient evidence to show that Graves's disease is not a primary thyroid disease but depends upon the same constitutional anomaly or defect which renders the control of visceral functions through the autonomic nervous system inadequate.

The protean clinical manifestations of Graves's disease are more easily explained as dependent on variations in the activity of the visceral nervous system rather than that they are dependent upon either qualitative or quantitative changes in the thyroid secretion.

ABSTRACTS OF PAPERS DELIVERED AT SECTION MEETINGS

Section of Orthopedic Surgery, April 15, 1927

FRACTURE OF THE FIFTH METATARSAL BONE WITH SPECIAL REFERENCE TO DELAYED UNION

LOUIS CARP

Twenty-one cases of fracture of the fifth metatarsal bone will be considered to illustrate a tendency toward delayed union probably caused by poor blood supply. Knowledge of this tendency is necessary for prognosis and treatment.

In 1855 Breithaupt first described the condition known as "Fussgeschwulst." He noted that soldiers on the march were frequently disabled by painful, swollen and tender feet, and he attributed this condition to strained ligaments and tendons. Subsequently the condition was described under various other names, but in 1898 Kirchner first showed it to be a fracture by roentgenogram. There have been occasional references in the literature to slow formation of callus in some fractured metatarsals. Delayed union in some fractures of the fifth metatarsal could easily explain the picture presented in "Fussgeschwulst."

The fifth metatarsal bone consists of the base, tuberosity, shaft and head. It must be remembered that the tuberosity has a separate epiphysis which makes its appearance at about the twelfth year and is completely ossified at about the sixteenth. The muscle and ligamentous attachments to the base and the shaft play an important rôle in the production of fracture by indirect violence. The nutrient vessel of the fifth metatarsal is very small and is represented only by a few fine branches which anastomose with the small blood-vessels of the epiphyses.

The usual mechanism of fracture of this bone is by indirect violence, while its exposed position is a predisposing factor for its injury by direct violence.

An analysis of twenty-one cases from the Presbyterian Hospital given in the table shows the following:

1. Sex { Males 9 (47%); Females 12 (53%)

2. Age { Youngest 13 years; Oldest 60 years; Average 36 years

		Base	Tuberosity	Shaft	Distal extremity	Total
3. Violence	Direct	3 (38%)	1 (12%)	2 (25%)	2 (25%)	8
	Indirect	7 (64%)	2 (18%)	1 (9%)	1 (9%)	11
	Doubtful	2 (100%)				2

4. Predominant symptoms and signs—pain, swelling, ecchymosis and tenderness.

5. Length Disability (16 cases) { Shortest 2 weeks; Longest 24 weeks; Average 10 weeks

6. Females tend to have a longer disability than males.
7. The end results are good.

A further analysis of the twenty cases in adults shows that there were five cases which from clinical and X-ray evidence had delayed union. This occurred four times at the base and once at the distal extremity of the shaft. All the other cases that could be followed had characteristic pain, tenderness and edema at and surrounding the area of fracture, which extended over periods of weeks or months. While it is true that soft part injury in association with the fracture might produce these symptoms for a short period, still interference with proper bony union is the most plausible explanation for the long disability.

One would expect in a long bone as small as the fifth metatarsal that enough union would take place in the cancellous portions in about ten days to prevent mobility of the fragments. In the cortical bone of the shaft normal calcification should occur in about three weeks. The callus may be scarcely visible in the X-ray, it may be excessive, or it may be long and thin. It must be remembered, however, that the time for the appearance of callus in the X-ray is variable.

The treatment recommended for fracture of the fifth metatarsal is immobilization of the foot and leg by means of a posterior moulded plaster splint, which will permit physiotherapy. If there is a tendency to delayed union, the administration of cal-

cium or cod liver oil is recommended. Scarification of the fractured end with a needle introduced through the soft parts in order to produce bleeding may be tried. Excessive trauma to demonstrate mobility of the fragments is to be avoided.

Summary

Twenty-one cases of fracture of the fifth metatarsal are reported, with their analysis. Twenty were in adults, and of these five showed clinical and X-ray evidence of delayed union. All the latter had a normal blood calcium and phosphorous and a negative urine and blood Wassermann. All the other cases that could be followed had clinical symptoms over such long periods that it is fair to assume some interference in bone repair. The main cause of this condition is probably the poor blood supply of this bone. The treatment is directed toward immediate immobilization and hyperemia. The experience from all these cases tends to show that too long immobilization produces bone atrophy which certainly cannot help bone repair. Weight bearing in a strong moulded plaster splint before one month, where possible, is suggested to overcome this bone atrophy. Between the 11th and 16th years, the epiphysis of the tuberosity is not to be mistaken for fracture.

Section of Ophthalmology, April 18, 1927

THE CULTIVATION OF LENS EPITHELIUM*

DANIEL B. KIRBY

It was proposed by Dr. John M. Wheeler that an original investigation be conducted into the nature of the cause of senile cataract and the possibility of its prevention and arrest by non-surgical means.

It was decided to approach the problem from a scientific rather than an empiric standpoint and to go back to fundamentals. A study of the developing lens in the living embryo and of the

* The reader is referred to the complete article in *Transactions* of the American Academy of Ophthalmology and Oto-Larnygology, 1926, page 136.

living cells of the lens offered possibilities. It was also thought that if the cells of the lens could be cultivated outside the body that here would be a new tool, a new standard for our investigation into the nutrition of the lens. If the study of the cause of cataract could be narrowed down to the life and death of a single cell derived from the lens, a great step forward would be taken. The single cell is not so simple, but it is far simpler than the complex lens organ.

Harrison discovered in 1907 that cells could be caused to migrate and divide outside the body. In 1910, Carrel devised an ingenious technique by which the method of tissue culture is becoming adapted to its main object, the study of the fundamental problems of physiology and pathology.

Conclusions

By a series of experiments, the author proved that:

1. It is possible to dissect out the lens of a five-day-old embryo chick in such a manner as to free it from all extraneous cells.
2. Lens epithelium, even at a very early stage, is contained within a capsule.
3. Lens epithelium, under proper conditions, will live, migrate, divide and multiply outside the body in primary explants.
4. An unmixed strain of lens epithelium can be propagated from a primary explant by successive sub-cultures. This strain can be utilized for controlled experiments on the nutrition of the lens cells.

TREATMENT OF THE COMMONER SYPHILITIC LESIONS OF THE EYE

J. G. Hopkins

By the methods at present available it is possible that early cases of syphilis can be definitely cured. In a certain proportion of late cases also, the patient remains free from symptoms and negative in their reactions. It is therefore imperative to give all early and most late cases the treatment which offers the best prospect of a permanent arrest.

There is wide difference of informed opinion as to choice of drugs and duration of treatment, also as to whether treatment should be continuous or intermittent, and whether the drugs used should be given simultaneously or in alternating courses. The method of choice for early cases is continuous treatment for at least two years with arsphenamine or neo-arsphenamine, bismuth and mercury, in adequate dosage and in courses of one drug at a time. In gummatous lesions and most others of the tertiary type, potassium iodide is also of value.

Patients with iritis in the secondary period should invariably be treated intensively for two years. The governing consideration in their treatment should be not the healing of the eye lesions but the clearing-up of their general infection.

Patients with interstitial keratitis or tertiary iritis should be treated with some efficient preparation of arsphenamine and with iodides. The arsphenamine treatment should be given in courses of from six to ten injections alternating with courses of bismuth and mercury. Continuous treatment should be carried on for at least a year and intermittent treatment for two years further.

It is established that some cases of optic atrophy remain arrested for a long period after adequate arsphenamine treatment. Most of the cases which respond favorably are due to meningo-vascular syphilis, but some to tabes. The best results have been obtained from intraspinous treatment.

It is probable, also, that in other cases second nerve degeneration has been hastened by treatment. However, if the prognosis without treatment is absolutely bad, it seems advisable to attempt administration of some form of arsphenamine intravenously in all, and intradurally in those showing any abnormality in the cerebro-spinal fluid.

Section of Surgery, May 6, 1927

GASTRIC SECRETION AFTER SUBTOTAL GASTRECTOMY*

EUGENE KLEIN

Hydrochloric acid is one of the important factors in the production of peptic ulcers. The good results after partial gastrectomy have been ascribed to the production of an anacidity. Lewisohn has reported that 77 per cent. of the cases show no free acid after partial gastrectomy. Others have had similar results. The thought naturally occurs that if an anacidity was effected in 77 per cent., it would be of value to ascertain why the remaining 23 per cent. were not rendered anacid. In addition some gastrojejunal ulcers have recently been reported after this operation. It would be interesting to discover if these had occurred among the group in which the acidity had persisted. To study these questions a more careful analysis of the acid conditions was made after every partial gastrectomy. In place of the Ewald test meal which had previously been used, fractional (Rehfuss) test meals were made over a period of three hours.

A consideration of the physiology of the stomach shows that the operation of partial gastrectomy removes only one of the phases of gastric secretion.

The *primary* or psychic phase is due to impulses which reach the stomach over the vagi and are initiated chiefly by the tasting and chewing of food. This response varies in different individuals and also in the same individual at different times. It is directly proportional to the amount of appetite.

The *secondary* phase follows on the contact of the gastric mucosa with certain food and chemical substances, the most important being the products of protein digestion. Since the primary phase which initiates gastric digestion lasts about two hours, sufficient acid and ferment are secreted to digest some of the food present and therefore furnish these protein products.

* From the Surgical Service of Dr. A. A. Berg and from the Laboratory, Mount Sinai Hospital, New York City.

The acid and peptic cells are located in the body and fundus of the stomach. There are none in the antrum. Nevertheless, stimulation of the body and fundus by the presence of protein products *does not cause* gastric secretion. Similar stimulation of the antrum *does*. The site, therefore, for the secondary stimulus is the antrum. This is the part of the stomach removed in partial gastrectomy.

The intestinal phase of gastric secretion starts one to two hours after the entrance of food into the intestine. Its relative importance in man is not yet determined.

In addition to these there is the so-called continuous secretion. It is probable that the gastric glands are always active. But while in the majority of individuals the secretion, except following the above stimuli, is small, in others it may be very large. Though a continued secretion with a high acidity in a stomach that contains no food may be pathological, it is often found in apparently normal individuals. It may be that those with the so-called ulcer diathesis fall within this group, since this condition is very common in duodenal ulcer. The origin of this secretion is unknown. It may be due to an exaggerated vagus tone.

The amount of acid secreted in different people varies, since the stimuli exerted by these different phases are not of the same potency for all. Normal individuals thus fall into hypo, iso and hypersecretory groups, the largest being the middle or isosecretory group.

Partial gastrectomy removes the antrum and hence the secondary phase. This is ordinarily the most important. It, therefore, reduces quantitatively the amount of acid secreted. The remainder is neutralized wholly or in part by the food, saliva, bile and pancreatic juice. Whether it will be completely neutralized depends on the potency of the other phases in the particular individual. If, for instance, there is a very high and prolonged continuous secretion due to a markedly exaggerated vagus tone, antrectomy will only reduce the acidity. But where these phases are not overactive, antrectomy will produce a hypo or anacidity.

Table 1 shows the maximum acidity in the Rehfuss test meals immediately after (Recent), and six months after (Old) partial gastrectomy. These are compared to preoperative findings. In the group of duodenal ulcers there is a moderate immediate and

MAXIMUM FREE ACID AFTER PARTIAL GASTRECTOMY IN FRACTIONAL TEST MEALS

		Anacid	0–20	20–50	50+	No. of cases
Duodenal	Unoperated		4%	36%	60%	50
	Recent	9%	9%	46%	36%	11
	Old	25%	41%	17%	17%	12
Gastric	Unoperated		28%	60%	12%	25
	Recent	45%	33%	11%	11%	9
	Old	100%				3
Gastrojejunal	Unoperated		11%	67%	22%	9
	Recent	25%	25%	50%		4
	Old	50%		50%		2

RECENT refers to cases examined about three weeks after operation.
OLD refers to cases examined about six months after operation.
UNOPERATED in GASTROJEJUNAL group refers to cases examined before partial gastrectomy.

a marked late reduction. In the gastric ulcers there is a marked diminution in acidity both immediately and six months later. The same is true of the group of gastrojejunal ulcers, although the number of post-operative cases is still small. A high continuous secretion is common in duodenal ulcer. It is rare in gastric ulcer. It may be that this accounts for the higher acidity left in the duodenal group. The cause for the further reduction after six months cannot be given. Possibly this is due to a lowering of the vagus tone.

While partial gastrectomy does not apparently produce an anacidity in as large a percentage of cases as previously believed, it nevertheless accomplishes a far greater reduction than gastroenterostomy. Gastroenterostomy produces no quantitative diminution of acid secretion. It must hope to neutralize all of the acid secreted from all of the phases and it rarely succeeds.

That the amount of acid reduction actually achieved by partial gastrectomy is significant, is shown by the few recurrences reported after the operation. Where gastrojejunal ulcers do follow, it is no doubt due to the fact that a sufficient lowering of acidity was not effected because of the reasons mentioned above.

Whatever acid did remain was sufficient in combination with the other causative factors of ulcer to produce a recurrence.

Section of Neurology and Psychiatry, May 10, 1927

STATIC AND KINETIC INNERVATION

J. Feser

The whole brain mechanism can be divided into a mechanism for the static innervation and one of the kinetic innervation. Static innervation means posture, attitude; kinetic innervation means actual and virtual movements of the body, limbs, eye, tongue, etc.

The apparatus of static innervation is described by Spiegel, *Klinische Wochenschrift,* 1926, page 277, as follows:

First, it consists of the centers in the rhombencephalon, the nucleus Deiters and the cells of the *formatio reticularis,* which are the centers of the decerebrate rigidity. It is worth mentioning that a similar phenomenon in the frog was described before Sherrington by Verworn in his "General Physiology," 1898.

The centers for the prevalence of extensor tonus, the decerebrate rigidity, are balanced by the *nucleus ruber* with the rubrospinal tract, resulting in an even distribution of tone in the muscle system. The midbrain animal is able not only to stand, but also to move.

A further tonus-regulating influence issues from the cerebellum through the *brachium conjunctivum* to the *nucleus ruber* and through the *corpus restiforme* to the cells of the *formatio reticularis.*

The cerebellum, in its turn, is under a static influence from the brain cortex through the fronto- and temporo-pontine tracts.

Finally, the *nucleus ruber* is under the control of the striatum, which is *via* thalamus influenced by the brain cortex also.

Such is the apparatus of the static innervation as described by Spiegel.

The apparatus of the kinetic innervation, on the other hand, consists of the pyramidal tracts and their centers, the occipital,

parietal and temporal lobes with the exclusion, of course, of the sensory terminals in these regions.

Its function concerns the movements of the body and limbs, and also, according to the given anatomical delimitation, the intellectual behavior, language spoken and written, imagination, the formation of representations and actions. Thinking, it appears, is a dream of doing something, as H. Jackson aptly said.

The line thus drawn between a static and a kinetic innervation corresponds to a line we are very familiar with in psychology. Here the most fundamental and most general distinction to be made is the distinction of a "What" and a "That." In other words, there is a certain content, a "What," perceived or imagined, and there is our attitude towards it; a "That" by which we characterize the content, as agreeable or disagreeable, as well-known or hardly noticed, as familiar or strange, as willed or desired, etc. Attitude, the "That," is a function of the static apparatus; content, the "What," is a function of the kinetic, the intellectual apparatus.

Thus it is possible to reduce the psychological data to terms of behavior, to posture and movement, and to get rid of such obscure words as mind and consciousness.

The mental diseases can be classified on the basis of this distinction. Kleist, in a paper, "Die gegenwärtigen Strömungen in der Psychiatrie," has made a similar attempt when he divided psychiatric diseases into those predominantly of the brain-stem and those of the brain cortex.

Hypnotism and suggestion are put on a physiological basis; they are revealed as conditions where the individual does not or cannot adapt his attitude to his impressions. His attitude is bound, as his muscles are bound. This seems the simple truth from the standpoint of motility.

Finally, neurasthenic and hysterical reactions present themselves obviously as diseases of the brain-stem and of perhaps the basal ganglia; they are postural, attitudinal disorders.

Thus it appears that the facts of psychology and psychopathology, or better, of normal and abnormal behavior, receive a new light from the distinction of static and kinetic innervation.

They are put on an intelligible, neurological basis, from which further investigation concerning their pathological nature can start.

Freud, in one of his latest papers, "Die Laienpsychoanalyse," says: "For the psychoanalyst the neuroses are an annoyance and embarrassment (ein Aergernis und eine Verlegenheit)."

This applies truly to any attempt to attack these conditions on purely psychological grounds.

A more favorable attack can be made when neuroses and psychoses are understood from the standpoint of motility, consisting of its two factors, posture and movement, static and kinetic innervation in their mutual interrelation.

Section of Otology, May 13, 1927

MASTOIDITIS IN INFANTS

ARTHUR M. ALDEN, St. Louis, Mo.

Prior to about two years ago a surgical mastoiditis in an infant was rarely diagnosed except upon the basis of external signs; redness, swelling or subperiosteal abscess. Largely as the result of work done in the St. Louis Children's Hospital and followed up and corroborated elsewhere, we now know that an infant may have a very severe type of mastoid infection capable of producing profound constitutional symptoms and even death without any of the conventional signs of mastoiditis being present.

Routine autopsies on infants who had died as a result of what has hitherto been called cholera infantum, marasmus, or intestinal decomposition have uniformly failed to show causal pathology in the gastro-intestinal tract, but almost without exception pronounced infections of the mastoid antra and middle ears were demonstrated.

Mastoiditis in adults has been classified from a pathological standpoint into two types, coalescent and hemorrhagic. In the former, the local spread of the infection is by direct continuity and the symptoms are, for the most part, the results of pressure.

In the latter, the dissemination of the infective agent takes place by way of the local blood or lymph channels and the symptoms are the result of massive and profound toxic absorption. Each of these types of mastoiditis in the adult has its analogue in the infant. The first type includes the hitherto-accepted classic form of mastoiditis with its local swelling, redness and subperiosteal abscess. The diagnosis in this type of the disease presents no difficulty and is often made by the parent. In the other type of the infection, which in the infant we have called the non-coalescent type of mastoiditis, external signs are as a rule absent and the predominating symptoms are fever, diarrhea and vomiting, which are the result of septic absorption from the infected ears. The diagnosis as to the true cause of this gastro-intestinal syndrome is often missed because the ears of these cases are not carefully examined. The otologic signs in this latter type are change in color and luster of the ear drum to a dirty gray or yellowish appearance. Rednesss and bulging may or may not be present. A sag in the superior wall of the canal external to the drum is pathognomonic of this condition. Such drums should always be incised, and incision is usually followed by a prompt remission of the intestinal symptoms.

The pediatric treatment of this type of case is very important and consists of supportive therapy in the nature of fluids, Ringer's solution and transfusions, usually administered directly into the superior longitudinal sinus. Only, when in spite of adequate drainage through the ear drums and all supportive measures, the condition of the child as shown by the weight and fever curve becomes progressively worse, do we feel that external drainage of the mastoid antra must be done. Earlier in our work we were reluctant to operate upon these babies until they were almost moribund, and many of our early fatalities could, I am sure, have been prevented had they been operated earlier in the course of the disease. When it is determined that mastoid antrotomy is indicated, the sooner that it is done the better will be the prognosis for the little patient, because these babies often pass from a very fair to an almost dying condition in a few hours.

In our hands all the operations have been carried out under local anesthesia. For this purpose, one-half per cent. novocaine with ten drops of one to one thousand adrenalin to the ounce has

been used with very satisfactory results. The skin over the mastoid and the periosteum covering the mastoid antrum are thoroughly infiltrated. The incision required is usually one and one-half to two cm. in length. The only landmark which it is necessary to see is the posterior-superior margin of the external auditory canal or the posterior-superior margin of the annulus. The infantile mastoid antrum lies just behind and above this point. After the periosteum is elevated, the cortex is removed by a rotary motion of an eight mm. Alexander gouge. This usually completely unroofs the tiny mastoid antrum. All overhanging edges are removed with forceps and the granulations gently wiped or curetted from the mastoid cell. The curette should never be passed forward into the aditus or attic on account of the danger of tearing or displacing the ossicular attachments. When the operation is finished the antrum is lightly packed with a gauze drain and no attempt at surgical closure of the edges of the incision made.

The first dressing is usually changed on the second day after operation, and from that time until the wounds are healed daily dressings should be the rule. Sterile dressing technique is very important, because in those cases which become secondarily infected the convalescence is, as a rule, prolonged and stormy. The gauze drain should be kept in place until inspection of the ear shows the tympanic membrane to be closed or the perforation, if patent, dry. In those cases which show an obstructive mass of adenoids or a nasopharyngitis which does not yield readily to treatment, an adenoidectomy performed before the child leaves the hospital will probably help to prevent recurrence.

In two and one-half years' experience with these cases we have learned many things. At first, cases were operated upon only when disaster threatened. Of the first seventeen cases which came to operation, we were able to save nine. Realizing that some of the cases which died could probably have been saved had they been operated earlier, we became somewhat bolder, and of the next nine cases only three died. In the next series of forty cases we saved all but five, two dying of pneumonia, one of meningitis, one of athrepsia and one of septicemia.

Lantern slide demonstration of cases, temperature and weight charts and technique of the operative procedure.

Section of Obstetrics and Gynecology, May 31, 1927

THE NASAL APPLICATION OF PITUITARY EXTRACT FOR OBSTETRICAL PURPOSES

J. HOFBAUER, Baltimore

The recent clinical study of Scott on the present state of induction of labor revealed the fact that, up to this time, there is no method of serving this purpose that is absolutely free from danger to either mother or child, and more particularly Watson's technique has some dangers to the infant, due, for the most part, to the occasional tetanic contractions which sometimes follow the use of the drug. Watson's method is based on the administration of castor oil and quinine with the object of sensitizing the uterine muscle, followed by the repeated subcutaneous injection of 0.5 cc. doses of pituitary extract at half-hour intervals until labor sets in or until six doses have been given. Those who have had experience with this method agree that too strong an action of pituitrin may produce disastrous effects on the child; furthermore, since the presence of toxemia or cardiac complication is generally considered a distinct contra-indication, the method should be employed only in selected cases.

The general recognition of these shortcomings of Watson's method supplied the impetus for us to seek some method of improving it. Two main considerations influenced our search for new channels through which pituitary extract may be absorbed in a more effective and at the same time in a less dangerous way. The desideration being to discover, if possible, whether the local application of pituitary extract to some mucous membrane would permit its *slow* and *steady* absorption. The possibility of withdrawing the drug from the mucous membrane as soon as the tendency of the uterus to pass into tetanic contraction became noticeable, was considered an essential feature of any attempt to replace the hypodermic route.

The oral administration of pituitary extract, both for the purpose of inducing labor and of stimulating pains in cases of *inertia uteri*, was first considered, since under experimental conditions a marked effect upon the uterus, increasing both its tone and auto-

matic contractions after administration of this drug to the sublingual region, was clearly evidenced by the recent work of Knaus. We employed this technique for the induction of labor in twenty patients. Labor was successfully induced in nine out of these twenty patients—an efficacy of 45 per cent. results. In eleven additional instances the same technique was employed to stimulate pains in patients who were already in labor but in whom the pains had stopped for a number of hours—irrespective of the degree of cervical dilatation and regardless of whether the membranes were ruptured or not. In ten of these cases a satisfactory result was obtained without ill-effect to either mother or child.

In the search for another portal of entry from which the pituitary extract could be absorbed more satisfactorily, our attention was directed especially to the nasal route, taking particularly into account the consideration that the mucous membranes covering the inferior turbinate constitute a highly vascular tissue whose vascularity and tendency to engorgement, as well as the width of its lymphatic beds, are markedly increased during pregnancy. Since, in the present paper, we propose to deal chiefly with the therapeutic utilization of the nasal mucosa for the absorption of pituitary extract, both by blood stream and by lymphatics, an accompanying semi-diagrammatic representation of the lateral nasal wall may be of service for a ready understanding of the topography. Under direct vision, with the aid of a nasal speculum and reflected light, a small pledge of cotton of such size as to fit easily but snugly between the septum and the inferior turbinate, and moistened with 10–20 minims of pituitary extract, is inserted under the anterior end of the inferior turbinate. If the pledget is inserted gently the patient does not experience any appreciable discomfort, and in none of our cases was it necessary to resort to any kind of local anesthetic. In every instance prior to the nasal application of pituitary extract the patient was prepared in the routine manner of giving a hot drink and castor oil and quinine by mouth, followed by a high enema. A period of two to three hours was allowed to elapse between the preliminary preparation and the administration of the first dose of pituitrin in order to make sure that the former had not been sufficient to induce pains. Uterine contractions invariably follow the nasal application of pituitary extract

within one to five minutes. In the event that the first contraction lasted longer than four minutes, the pledget was withdrawn and the uterus relaxed within the next 60–70 seconds. If no tetanic contraction occurred during the first fifteen minutes, we considered that the danger from such an accident had passed.

Unless the first application has resulted in setting up satisfactory contractions at the end of two hours, the pledget is withdrawn and a fresh one applied to the opposite nostril for a similar period of time. In the majority of our cases one to three applications were required for a successful induction of labor. In only a few cases five doses were given. It is important to bear in mind that our experience shows that in pre-eclamptic cases labor can be induced with the greatest ease, one application of pituitrin usually being sufficient; whereas, greater difficulty was encountered in the nephritic cases in which the response was definitely less pronounced. It is interesting to note that in none of the toxemic cases did the blood pressure rise materially over the initial level during the course of induction. Furthermore, we came to realize that while the method under consideration is rarely effective in normal pregnancy much before term, it gives satisfactory results in toxic patients a month or six weeks before term.

Up to the present time we have employed the nasal method of inducing labor in seventy patients. The indications for the induction of labor are given in the accompanying table:

Toxemia	28 cases
Postmaturity	12 "
Severe pyelitis	5 "
Hydramnios	2 "
Dead foetus	2 "
To test the method in normal cases at term and in the last month of pregnancy	21 "

In group 1–5 (forty-nine cases), labor was induced successfully in all cases recorded. In the last group nine failures occurred. We found here that patients in whom the cervix still presented a length of 2 cm., with the external os tightly closed, offered the least chance of a good result, while those showing obliteration of the cervical canal offered much better prospects. This observation is quite in harmony with the results of recent experiments

of Knaus, which indicate that considerable changes occur in the relationship between the hypophysis and the uterine muscle during pregnancy, and that, due to an increase of the contractability and the tone of the uterine muscle toward the end of pregnancy, labor can be induced by comparatively small doses of pituitrin at term but not before term.

The fact that in our series all of the babies have been born alive testifies to the control we possess over the action of the drug. Furthermore, it may be mentioned that we have also found the nasal method a safe and efficient procedure in accelerating labor when already in progress. In conclusion, our investigations also show that the administration of repeated small doses of pituitary extract renders a valuable service in cases of pyelitis in pregnancy.

PRELIMINARY ANNOUNCEMENT OF STATED MEETINGS OF THE ACADEMY

1927–1928

October 6.—*The economic cost of disease.* Edgar Sydenstricker, Statistician, U. S. Public Health Service, Washington; Louis I. Dublin, Statistician, Metropolitan Life Insurance Company of New York; Leland E. Cofer, Director, Bureau of Industrial Hygiene, State of New York.

October 20.—*Carpenter Lecture:* The present and future food supply of the United States. Alonzo E. Taylor, Director of Food Research, Stanford University, California.

November 3.—*Recent knowledge of epidemic diseases.* Leslie T. Webster, Rockefeller Institute for Medical Research, New York.

November 17.—*Anniversary Discourse.* John Dewey, Professor of Philosophy, Columbia University, New York.

December 1.—*Malaria in syphilis.* George H. Kirby, Director Psychiatric Institute of the New York State Hospitals, and Henry A. Bunker, Assistant Director; Charles W. Stone, Associate Professor of Nervous Diseases, Western Reserve University, Cleveland.

December 15.—*Experimental work on the tumor question.* James B. Murphy, Rockefeller Institute for Medical Research, New York.

January 5.—*Annual Meeting.* On the significance of bacterial allergy in infecticus diseases. Hans Zinsser, Professor of Bacteriology and Immunology, Harvard University.

January 19.—*Graduate medical education.* Louis B. Wilson, Director Mayo Foundation, Rochester, Minnesota.

February 2.—*Alcoholism.* Matthias Nicoll, Jr., Commissioner of Health, State of New York; Charles Norris, Chief Medical Examiner, City of New York; Alexander O. Gettler, Assistant Medical Examiner, City of New York.

February 16.—*Animal diseases in man.* Theobald Smith, Director Department of Animal Pathology, Rockefeller Institute, Princeton.

March 1.—*The eye in relation to disease.* G. E. deSchweinitz, Philadelphia.

March 19.—*Physical therapy.* Frank B. Granger, Boston.

April 5.—*Management of the psychoneuroses.* Austin F. Riggs, Stockbridge, Mass.

April 19.—*The future of surgery.* Walton Martin, New York.

May 5.—*Behaviorism and delinquency.* William Healy, Director Judge Baker Foundation, Boston; John B. Watson, New York.

May 17.—*Recent biological studies and their significance.* Edmund V. Cowdry, Rockefeller Institute for Medical Research, New York.

RESOLUTION REGARDING THE VOLSTEAD ACT

Passed at the Stated Meeting of the Academy, May 5, 1925

Whereas, the Federal Law, known as the Volstead Act, in the provisions of Section 7, the constitutionality of which has been upheld in a recent decision of the Supreme Court of the United States, limits the freedom of the physician in prescribing for his patients; and,

Whereas, this Congressional assumption of power belonging to the States takes away from the States their right to regulate the practice of medicine within their own borders; and,

Whereas, this power may be extended so as to limit the use of other drugs for the satisfactory and scientific practice of medicine and to the discomfiture of the States; be it

Resolved, that The New York Academy of Medicine hereby records its opinion that Section 7 of the Volstead Act is a serious infringement upon the legitimate practice of medicine, and that the Council of the Academy is hereby authorized to take such steps as it may deem necessary to secure a modification of this Section so that there will be no limitation in the time during which one pint of spirituous liquor may be administered under due regulations to prevent the illegal use of spirituous liquor for beverage purposes.

COMMITTEE ON MEDICAL EDUCATION

Opportunities for Graduate Medical Study in Hospitals of New York City

The Committee has just published a revision of the booklet entitled, "Opportunities for Graduate Medical Study in Hospitals of New York City," the first edition of which appeared about a year ago. Besides a re-arrangement of its contents, considerable new matter has been added, so that the booklet now presents in concise form a fairly accurate description of the many and varied opportunities which are offered for clinical study in the hospitals of greater New York.

The main portion of the booklet is devoted to details describing the special opportunities which are offered in each of the ninety teaching hospitals, with the names of the clinicians and the hours when the clinics, conferences, grand rounds, etc., are held.

A list of more than 230 special internships or residencies in the clinical specialties offered in the hospitals of the city is presented for the first time. There are also lists of undergraduate and postgraduate medical schools and of the graduate medical courses offered in hospitals of the city.

The booklet has been prepared particularly for the use of medical visitors whose stay in the city is limited. Copies may be obtained on application to the Bureau of Clinical Information.

INDUSTRIAL HYGIENE EXHIBIT AT THE ACADEMY

Beginning on Thursday, October 6th, and lasting for about ten days, there will be at The New York Academy of Medicine the Industrial Hygiene Exhibit of the New York State Department of Labor, comprising wax models illustrating some of the most important industrial diseases; a collection of dusts which are the natural by-products of many of the industries; glass transparencies showing the effects of injurious dusts upon the body; various devices used in the study of the causation of industrial diseases; working models showing mechanical devices for removing dusts from work rooms, and moving pictures showing various conditions in industry and the application of Industrial Hygiene to the general betterment of these conditions.

CANDIDATES RECOMMENDED FOR ELECTION TO FELLOWSHIP OCTOBER 6, 1927:

Irving Balensweig, M.D., 34 West 91st Street.
Milton Arlanden Bridges, M.D., 148 West 74th Street.
Lyman Weeks Crossman, M.D., 13 East 11th Street.
George William Cumbler, M.D., 65 East 55th Street.
Arthur Christian DeGraff, M.D., 142 East 33rd Street.
Connie Myers Guion, M.D., 147 East 50th Street.
Abraham Kardiner, M.D., 1150 Fifth Avenue.
Harry Clifton Luke, M.D., 121 East 60th Street.
Frank Joseph McGowan, Jr., M.D., 100 East 66th Street.
John James McGowan, M.D., 472 West 143rd Street.
Frederick Westcott Solley, M.D., 114 East 60th Street.
Samuel Stern, M.D., 40 East 51st Street.
Arthur Hutchinson Terry, Jr., M.D., 137 East 66th Street.
Grant Thorburn, M.D., 379 Park Avenue.
Seymour Frederick Wilhelm, M.D., 4 East 95th Street.

RECENT ACCESSIONS TO THE LIBRARY

de Abreu, M. Essai sur une nouvelle radiologie vasculaire.
Paris, Masson, 1926, 215 p.

Argelander, A. Das Farbenhören.
Jena, Fischer, 1927, 172 p.

Armstrong, H. E. Essays on the art and principles of chemistry.
New York, Macmillan, 1927, 276 p.

Ashhurst, A. P. C. Surgery. 3. ed.
Philadelphia, Lea, 1927, 1179 p.

Berg, L. S. Nomogenesis, or evolution determined by law.
London, Constable, 1926, 477 p.

Bodin, J. Contre Freud. Critique de toute psychologie de l'inconscient.
Paris, Masson, 1926, 99 p.

Bondi, S. Herzhinterwand und oesophageale Auskultation.
Wien, Springer, 1927, 114 p.

Bretonneau, P. Die Diphtherie.
Berlin, Springer, 1927, 173 p.

Burnet, J. Diseases of the newborn.
London, Oxford Pr., 1927, 275 p.

Cemach, A. L. Das Problem der Mittelohrtuberkulose.
Berlin, Urban, 1926, 224 p.

Chiray, M. & Lebon, J. Les insuffisances pancréatiques.
Paris, Masson, 1926, 210 p.

Clark, A. J. Comparative physiology of the heart.
Cambridge, Univ. Pr., 1927, 157 p.

Coffey, W. B., Brown, P. K. & Humber, J. D. Angina pectoris.
New Orleans, Dickerson, 1927, 393 p.

Conduct (The) of medical practice. By the editor of "The Lancet" and expert collaborators.
London, "The Lancet," 1927, 332 p.

Cooley, E. J. Probation and delinquency.
New York, Nelson, 1927, 544 p.

Dubreuil-Chambardel, L. Variations des artères du membre supérieur . . .
Paris, Masson, 1926, 256 p.

Duchange, R. L'anesthésie tronculaire des machoires par voie buccale.
Paris, Masson, 1926, 112 p.

Ellis, H. The task of social hygiene. 2. ed.
London, Constable, 1927, 414 p.

Enzyklopädie der mikroskopischen Technik. 3. Aufl. Hrsg. von R. Krause.
Berlin, Urban, 1926–27, 3 vols.

Frieboes, W. Atlas der Haut- und Geschlechtskrankheiten, Lfg. 1.
Leipzig, Vogel, 1927.

Girel, C. La roentgenthérapie des épitheliomas cutanés . . .
Paris, Masson, 1926, 300 p.

Goldschmidt, R. Physiologische Theorie der Vererbung.
Berlin, Springer, 1927, 247 p.

Great Britain. Air ministry. Manual for medical officers of the Royal air force.
London, H. M. S. off., 1927, 241 p.

Greene, J. S. & Wells, E. J. The cause and cure of speech disorders.
New York, Macmillan, 1927, 458 p.

Grenet, H., Levant, R. & Pellissier, L. Les syphilis viscérales tardives.
Paris, Masson, 1927, 378 p.

Guillain, G., Larcohe, G. & Lechelle, P. Technique de la réaction du benjoin colloïdal.
Paris, Masson, 1926, 36 p.

Handbuch der praktischen Therapie. Hrsg. von R. von der Velden und P. Wolff.
Leipzig, Barth, 1927, 2v.

Harrower, H. R. The hepatic principle anabolin, detoxication by the liver and the control of functional hypertension.
London, Baillière, 1927, 147 p.

Hebert, G. T. Pulmonary tuberculosis.
London, Arnold, 1927, 212 p.

Henderson, D. K. & Gillespie, R. D. A textbook of psychiatry.
London, Oxford Pr., 1927, 520 p.

Hérail, J. Traité de maitière médicale pharmacographie. 3. éd.
Paris, Baillière, 1927, 819 p.

Hering, H. E. Die Karotissinusreflexe auf Herz und Gefässe.
Dresden, Steinkopff, 1927, 150 p.

Jones, P. C. V. The significance of temperature variations in tuberculous disease.
Cambridge, Papworth [1926], 174 p.

Ker's manual of fevers. Revised by C. Rundle. 3. ed.
London, Oxford Pr., 1927, 346 p.

Kleinschmidt, H. Tuberkulose der Kinder. 2. Aufl.
Leipzig, Barth, 1927, 221 p.

Knowles, R. & Senior-White, R. Malaria.
Calcutta, Thacker, 1927, 220 p.

Lehrbuch der speziellen Chirurgie. Hrsg. von J. Hochenegg und E. Payr. 2. Aufl. vol. 1.
Berlin, Urban, 1927.

Léri, A. Études sur les affections des os et des articulations.
Paris, Masson, 1926, 460 p.

McFall, R. J. The world's meat.
New York, Appleton, 1927, 624 p.

McNalty, A. S. Epidemic diseases of the central nervous system.
London, Faber, 1927, 194 p.

Monchamps, E. & Moritz, E. Les étapes mentales de l'observation des images.
Bruxelles, Oeuvre nat. de l'enfance, 1927, 172 p.

Müller, M. Johannes Müller.
Leipzig, Barth, 1927, 101 p.

Myers, J. A. The normal chest of the adult and the child.
Baltimore, Williams, 1927, 419 p.

Nové-Josserand, G. & Tavernier, L. Tumeurs malignes des os.
Paris, G. Doin, 1927, 424 p.

Parry, L. A. Some famous medical trials.
London, Churchill, 1927, 326 p.

Pearce, E. C. A textbook of orthopaedic nursing.
London, Sci. Pr., 1927, 155 p.

Pinch, A. E. H. Superficial radium therapy.
London, Radium Inst., 1927, 64 p.

Piney, A. Recent advances in haematology.
Philadelphia, Blakiston, 1927, 276 p.

Portmann, G. & Retrouvey, H. Le cancer du nez . . .
Paris, G. Doin, 1927, 973 p.

Renaud, M. Les cancers et leurs complications.
Paris, Masson, 1927, 322 p.

Ritchie, M. B. H. Aesculapius armaque.
London, Bale, 1927, 112 p.
Roche, A. E. Pyelography.
London, Lewis, 1927, 118 p.
Rose, M. S. The foundations of nutrition.
New York, Macmillan, 1927, 501 p.
Rudolf, G. de M. Therapeutic malaria.
London, Oxford Pr., 1927, 223 p.
Sargent, D. A. An autobiography.
Philadelphia, Lea, 1927, 221 p.
Sarton, G. Introduction to the history of science. v. 1.
Baltimore, Williams, 1927.
Sinclair, M. The Thomas splint.
London, Oxford Pr., 1927, 168 p.
Stengel, A. & Fox, H. A textbook of pathology. 8. ed.
Philadelphia, Saunders, 1927, 1138 p.
Stewart, F. H. Segregation and autogamy in bacteria.
London, Adlard, 1927, 104 p.
Strickler, A. The skin: its care and treatment.
New York, Appleton, 1927, 194 p.
Theobald, G. W. Normal midwifery.
London, Oxford Pr., 1927, 258 p.
Thomson, J. A. Towards health.
London, Methuen, 1927, 242 p.
Tipper, E. H. The cradle of the world and cancer: a disease of civilization.
London, Murray, 1927, 142 p.
Tracy, E. A. The white spots of epilepsy.
Boston, Howard, [1926], 129 p.
Transactions of the American laryngological association, 1926.
Truc, H. Hygiène oculaire et inspection du travail.
Paris, Masson, 1926, 183 p.
Williams, J. F. The principles of physical education.
Philadelphia, Saunders, 1927, 481 p.
Woodbury, R. M. Workers' health and safety.
New York, Macmillan, 1927, 207 p.
Worcester, A. Nurses and nursing.
Cambridge, Harvard Univ. Pr., 1927, 173 p.

NOTE

At the time of the opening of the new building last November an exhibition of "Early and Later Medical Americana" was arranged by the Exhibits Committee. A catalogue of this, containing twenty illustrative plates and many biographical and bibliographical notes, has now been printed. Those who are interested may obtain a copy in the Library.

CORRECTION

In the note in the Richard Bright Exhibit which appeared in the August number of the BULLETIN a mistake was made in a date, which should be 1814 instead of 1914, next to the last line, page 545.

DEATHS OF FELLOWS OF THE ACADEMY

JOHN COVERT BOYD, M.D., U. S. Navy; graduated in medicine from the Medical Department, New York University in 1872; elected a Fellow of the Academy, April 2, 1896; died, July 7, 1927.

THEODORE BAME BARRINGER, JR., B.S. (1894), M.D., 112 East 76th Street, New York City; graduated in medicine from the Medical Department, New York University in 1897; elected a Fellow of the Academy, January 4, 1906; died, July 15, 1927. Dr. Barringer was a Fellow of the American Medical Association, a member of the Alumni Association of New York Hospital and associate attending physician to the New York Hospital.

ELWIN WALLACE HANNOCK, M.D., 363 State Street, Albany, N. Y.; graduated in medicine from the Albany Medical College in 1910; elected a Fellow of the Academy, January 15, 1914; died, August 5, 1927, at Lake Springs, N. J. Dr. Hannock was attending orthopedic surgeon to the Memorial Hospital, Albany.

THOMAS WILLIAM SALMON, M.D., 123 East 53rd Street, New York City; graduated in medicine from the Albany Medical College in 1899; elected a Fellow of the Academy, April 6, 1916; died, August 13, 1927. Dr. Salmon was a Fellow of the American Medical Association, a member of the American Neurological Association, president of the New York Psychiatric Society and the American Psychiatric Association, a member of the American Psychopathic Society, and a member of the American Society for the Advancement of Science. He was, since 1921, professor of psychiatry at Columbia University, College of Physicians and Surgeons; senior consultant in neuropsychiatry in the American Expeditionary Forces during the World War; was awarded the Distinguished Service Medal; Brigadier General, Medical Officers' Reserve Corps; member of the medical council of the U. S. Veterans' Bureau and of the Permanent Interallied Committee on the After-Care of Disabled Soldiers; one of the editors of the Medical History of the World War; author of chapters on nervous and mental diseases in several textbooks, and a frequent contributor to medical journals.

CHARLES CARTLIDGE GODFREY, M.D., 340 State Street, Bridgeport, Conn.; graduated in medicine from the Dartmouth Medical College in 1883; elected a Fellow of the Academy, April 2, 1896; died, August 31, 1927.

1927

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